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Tuberculosis of the Cervical Lymph Nodes

STANLEY L. BLUMENTHAL, M.D.*

Tuberculous infection characteristically drains from a primary focus into regional lymphatic nodes. The lymphatic nodes which are initially involved often pass on the tubercle bacilli to other nodes. Tubercle bacilli also are commonly spread throughout the body in small numbers via the blood vessels in a post primary phase of dissemination.[1] Some of these organisms end their travels in lymph nodes. Each of these mechanisms plays a role in the development of tuberculous adenitis.

PATHOGENESIS OF TUBERCULOUS CERVICAL ADENITIS

Tuberculous cervical adenitis is the commonest form of peripheral tuberculous adenitis.[2] This is most likely because of a lowered resistance in the cervical nodes due to recurrent acute infections of the pharynx and tonsils. Organisms which reach these nodes from other lymphatic or hematogenous pathways often find conditions suitable for growth there.

The upper jugular nodes (tonsillar nodes, mandibular nodes) are the ones most frequently affected in tuberculous cervical adenitis. In a series of 370 cases of tuberculous cervical adenitis, Bailey[3] found that in 54 per cent the upper jugular-mandibular nodes were attached. There was involvement of the lateral cervical-posterior triangle group in 22 per cent, the submaxillary in 12 per cent, and the supraclavicular and other nodes in 12 per cent. The nodes of the lateral cervical-posterior triangle group drain the adenoid area; they also drain the axillary nodes which in turn drain the pleura. The anterior deep cervical nodes also drain the pleura but in some instances may drain lymphatic pathways from the upper jugular nodes. In the usual primary tuberculous infection of childhood the initial lesion is located in the periphery of the lung; from here organisms are carried toward the hilus of the lung into the bronchial, tracheo-bronchial, and para-tracheal nodes. These nodes may drain into the anterior deep cervical nodes.[4]

There is much controversy concerning whether the upper jugular-mandibular nodes are infected through a primary lesion in the tonsils or through lymphatic or hematogenous pathways. Bailey[3] has reported that the upper jugular-mandibular nodes are infected through the tonsils in the majority of cases. On the other hand, Muschenheim[5] does not believe that primary

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tonsillar infection is a very frequent cause of tuberculous cervical adenitis. Recent authors who believe that there is a high incidence of tuberculous infection of the tonsils in those cases involving the upper jugular-mandibular nodes include German,[6] Swenson and Small,[7] and Nelson.[8] It is clear from a review of the literature that the tonsils and adenoids may be affected although they appear perfectly normal to direct inspection. This diagnosis can only be established with certainty by culture, but pathological diagnosis may be established with reasonable certainty on examination of microscopic sections. Indeed, Boyd,[9] in 1947, reported that 5 per cent of the tonsils removed in Canada appeared tuberculous on pathological section.

A review of the available data concerning the importance of tonsil and adenoid involvement in tuberculosis leads to the following conclusions: 1) The tonsils and adenoids may be affected although they appear grossly normal; 2) the cervical nodes most commonly infected with tuberculosis drain the tonsils and adenoids; and 3) tonsils which may have been infected by the hematogenous route may then drain into the cervical nodes. Therefore, in any case of tuberculous infection of lymph nodes draining the tonsils and adenoids, these organs should also be considered potentially involved.

PATHOLOGY OF TUBERCULOUS CERVICAL ADENITIS

When tubercle bacilli are filtered out by a lymph node, there is a resultant acute inflammatory reaction. This may result in transitory tenderness and local heat, but if these manifestations are present, they soon disappear. The exudative phase is often followed by caseation necrosis and cold abscess formation. As the nodes are gradually destroyed, there may be a perilymphadenitis which binds a group of nodes together. Healing mechanisms include fibrosis, calcification and resolution. A firm fibrous capsule may limit the process to one node or a group of nodes. This fibrous capsule, however, may rupture and the pus may extend under the deep cervical fascia where it is not readily discovered. After many months the deep cervical fascia may then erode and the pus may spread under the superficial fascia resulting in a "collar-stud abscess."[3] In such cases the distance between the necrotic node and the pus in the superficial space may be as much as 6 to 8 inches. A tuberculous abscess may also occur in the skin; such an abscess may rupture and form a draining sinus.

Healing mechanisms may halt the progress of tuberculous adenitis at any stage. However, even when fibrosis and calcification have arrested a lesion in the node, organisms may lie dormant for many years. Indeed they may remain viable throughout the life of the patient. Such organisms may break through their restrictions at any time to cause spread of infection many years after the development of the initial adenopathy.[10]

DIAGNOSIS OF TUBERCULOUS CERVICAL ADENITIS

The diagnosis of tuberculous cervical adenitis is suspected in the presence of a subacute or chronic nontender swelling in the neck.[7] The swelling may have been tender at first, has been present for at least four to six weeks, and does not appear to be subsiding even though adequate antibacterial antibiotic treatment has been given. Lymph nodes which are at first discreet and freely movable may at times become matted together and attached to surrounding tissue. The tuberculin test is usually positive. There may be a history of contact with an individual with tuberculosis. An x-ray of the chest may sometimes show a definite or questionable lesion. Occasionally an x-ray of the cervical region will demonstrate calcium deposits. If an abscess is evident, aspiration may be done, and diagnosis established by culture and pathological examination.

Certain points should be emphasized in the differential diagnosis of swellings of the neck. The most common cause of enlargement of lymph nodes in the neck is an acute or recurrent respiratory infection involving the pharynx and tonsils. Such infections are often streptococcal in origin; some may be due to viruses, even though absolute proof of this has not been established. Very often such inflammatory masses of the neck may remain enlarged for a number of months before subsiding. In occasional cases, staphylococcal organisms of low virulence will produce an abscess with very little pain or tenderness and no fever.

Problems in diagnosis of tuberculous cervical adenitis occur frequently. McDonald and Weed[11] have analyzed a large number of cases in which errors of diagnosis had been made. They have divided these into three groups: 1) a group in which tuberculosis has been mistaken for Hodgkin's disease, sarcoid and pyogenic infections but tubercle bacilli have grown out on culture; 2) a group in which tuberculosis has been suspected clinically and/or on biopsy but culture has revealed brucellosis or coccidioidomycosis; and 3) a group in which tuberculosis has been suspected on biopsy but no etiological agent has been found on culture. Some of the other disorders which must be considered in the differential diagnosis of tuberculous cervical adenitis include: toxoplasmosis, listeriosis, leukemia, Hodgkin's disease, metastatic tumor, actinomycosis, coccidioidomycosis, branchial cleft cyst, carotid body tumor, salivary gland tumor, neurofibroma, lymphosarcoma, and lateral aberrant thyroid tissue.[12] Recent reports of a form of cervical adenitis which is caused by chromogenic, saprophytic nontuberculous acid-fast bacilli provide an additional problem in establishing a proper diagnosis.[13]

The difficulty which may be presented in the diagnosis of an enlargement in the neck has been very well summarized by Lahey.[12] In an excellent discussion of tumors of the neck he stated that, in his opinion, "the final dif-

ferentiation between tuberculous nodes of the neck, lymphosarcoma and the various other tumors of the neck, either single or multiple, can be made with certainty only by the removal of a node, and on the basis of the pathological report."

Although the clinical diagnosis of tuberculous cervical adenitis can often be established with reasonable certainty, definitive diagnosis frequently requires biopsy and culture.[11, 12] When biopsy is done, culture should be made for tubercle bacilli, brucella organisms, fungi, and common pyogenic organisms. Cultures should be done without delay and should be performed on fresh pathological material which has not yet been placed in formalin.

TREATMENT OF TUBERCULOUS CERVICAL ADENITIS

There is considerable controversy concerning the proper treatment of a patient with tuberculous cervical adenitis. One school of thought believes primarily in prolonged chemotherapy and minimizes the use of surgery;[5, 14] the other group stresses the importance of early surgery.[15] Both viewpoints have their merit, but a study of the available data leads one to the conclusion that a combined approach in therapy will offer the greatest benefit to the patient.

In at least 50 per cent of cases of tuberculous cervical adenitis, the patient will have other tuberculous lesions in the lungs or elsewhere in the body. These lesions are often evident, but in many cases are occult and cannot be clinically detected. Those who maintain that chemotherapy should be the principal therapy stress that the patient with tuberculous cervical adenitis has a generalized disease. Therefore, they advise that therapeutic efforts should not be concentrated on the local lesion, but that instead, a general therapeutic management for tuberculosis and prolonged chemotherapy should be the foundation of the treatment program.[16] In the past, occasional good results have been obtained with the use of streptomycin;[2] however, in many patients this drug has been unsatisfactory. Recent studies[14, 17] have shown very encouraging results with the use of isoniazid in tuberculous adenitis. One such study was reported by Butler[14] in 1956; in 23 patients with tuberculous adenitis, 21 showed a good response to isoniazid after an average treatment period of 18 months. The superiority of isoniazid over streptomycin is probably due to its greater penetrating power in the presence of caseous tissue and macrophage cells.

The first principle of therapy is certainty of diagnosis. A child with a large cervical node of undetermined etiology which has been present for four to six weeks should have every effort made to determine any evidence of tuberculous infection. Studies should be made of the tuberculin reaction, x-ray of the chest, and gastric washings; investigation of possible contacts among family and friends should be carried out. Careful diagnostic investi-

gation should be performed to eliminate the possibility of any other cause of swelling in the neck. During this time the child should be started on a course of antibiotics. Penicillin is the drug of choice; tetracycline may be given in conjunction with penicillin if response is slow. This drug therapy should be maintained for a minimum of two to three weeks. A subacute bacterial adenitis will often show some response to such therapy; failure to respond is of some diagnostic value.

Surgical Therapy

Even though some apparently good results have been obtained from the use of isoniazid in causing shrinkage of tuberculous nodes, surgical treatment does offer considerable benefit. It must be recognized that any treatment which can eradicate caseous or fibrotic tuberculous foci is of great value; viable organisms may remain in such foci for many years. It should be stressed that organisms which have been lying dormant in inactive or "healed" tuberculous foci may flare up years later to cause widespread tuberculous lesions.[10] In recent years it has become relatively commonplace to remove segments of a lung because of an extensive tuberculous lesion. This may be done even though there are other clinical or occult tuberculous lesions in the body. In the light of these trends in the present day surgical therapy of pulmonary tuberculosis, it is unreasonable to refuse to consider the removal of a dangerous peripheral tuberculous lesion. Such lesions can be removed with considerably less hazard than a segment of a lung.[18]

Much of the confusion could be prevented by a proper understanding of the objectives of surgical therapy for this disease. These are as follows: 1) to remove a dangerous peripheral lesion which could otherwise serve as a potential focus for future tuberculous complications; 2) to remove an existing or potential caseous focus; 3) to remove a sinus tract; and 4) to remove a node or group of nodes for biopsy so that a proper diagnosis might be made. When a biopsy is performed it should be a therapeutic one with removal of all easily accessible diseased lymph nodes.[19] Tuberculous nodes of the neck should, however, not be removed by a radical block dissection, a type of operation commonly used for the treatment of malignancy.[3, 7, 18] Instead, there should be excision of the lymph nodes only, with preservation of all normal tissue.[7, 19] To obtain the best results it is necessary to perform a careful dissection of all diseased nodes. Cold abscesses attached to underlying nodes should be excised. The surgeon should be skilled in neck dissection and should take special precautions to avoid injury to the following structures: inframandibular branch of the facial nerve, spinal accessory nerve, hypoglossal nerve and internal jugular vein.[7, 15] An incision which is made parallel to the natural creases of the neck will usually heal with the production of only a small scar.

Lester and Jones[19] reported a large series of surgically treated patients in 1956. In a group of 66 patients with tuberculous adenitis, 65 had involvement of the cervical nodes; 8 individuals had axillary adenitis and 1 patient had inguinal adenitis. An active lesion was observed to be limited to the lymph nodes in about 50 per cent of the cases. Those patients who presented evidence of active pulmonary tuberculosis were treated with anti-tuberculosis chemotherapy and bed rest until the lesion in the lung was under control. If the tuberculous nodes were fluctuant, an early operative procedure was done to prevent rupture of the inflammatory mass. Fifty-four patients were selected for surgical treatment. Follow-up six months after surgery revealed that excellent healing had occurred in every case. Lester and Jones are convinced that the recurrence of a diseased node or the development of a sinus is often due to failure to remove all of the diseased nodes at the time of operation.

In a child whose tuberculous node has become fluctuant, early surgical procedures are often necessary to prevent rupture of the node. If there is a small cold abscess in a node or mass of nodes, it will generally be advisable to excise the abscess and the underlying nodal mass.[15] An alternate procedure[17] is to aspirate the cold abscess and instill a streptokinase-streptodornase solution into the cavity. After the abscess has subsided, excision should be performed. If a large cold abscess develops, it should be incised and drained, and irrigations with streptokinase-streptodornase solution should subsequently be carried out twice daily for several days and then once daily. In addition, the abscess cavity should be packed with gauze soaked in a solution containing these enzymes, and the packing should be changed daily. The irrigating solution should contain 400 units of streptokinase and 500 units of streptodornase per milliliter; the packing solution should contain 2,000 units of streptokinase and 2,500 units of streptodornase per milliliter. After several weeks of this treatment, healthy granulation tissue usually appears at the base of the node. Within several more weeks the rest of the lesion will often heal through further growth of granulation tissue. Excess granulation tissue can be excised or cauterized with silver nitrate. If the wound does not appear to be adequately healed after six to eight weeks of treatment, surgical removal of the remainder of the lesion should be performed.

The treatment of patients with tuberculous cervical adenitis should aim to prevent the development of sinus tracts. The use of broad spectrum chemotherapy in association with surgical procedures is of value in preventing sinus tract formation due to secondary bacterial infection. If a sinus tract does develop, and if it shows no signs of healing by granulation, it should be removed by surgical excision.

Chemotherapy

If diagnostic study reveals definite evidence of tuberculosis, the child should be started on therapy with isoniazid and para-aminosalicylic acid (PAS).[20] Isoniazid should be given in a daily dosage of 5 to 7 mg. per Kg. of body weight in two or three divided doses. The recommended daily dosage of para-aminosalicylic acid is 0.5 Gm. per Kg. of body weight divided into three or four daily doses with a maximum of 12 Gm. daily.[21] If anorexia, nausea and vomiting occur, the dosage of this drug should be reduced. In those children who continue to show severe gastrointestinal symptoms the drug may be discontinued. Treatment with isoniazid should be maintained for a minimum of a year; para-aminosalicylic acid therapy, except in the occasional case in which this drug is discontinued, should also be given for at least one year.

FOLLOW-UP THERAPY

After three to six months of treatment with isoniazid and para-aminosalicylic acid, the status of the involved node is re-evaluated. If it has almost completely disappeared, further treatment should consist only of a prolonged course of isoniazid and para-aminosalicylic acid. If there is a considerable degree of residual enlargement, surgery should be performed.[4, 17]

Patients in whom tuberculous adenitis is associated with an active pulmonary lesion should receive antituberculosis chemotherapy and bed rest until the lesion in the lung is stable. When the patient is afebrile, his weight stationary or increasing, his chest x-ray showing no sign of activity, and after he has received at least three to six months of treatment, the diseased lymph nodes should be re-evaluated. If they have not completely subsided, they should be considered for surgical removal.[19] Following the excision of tuberculous lymph nodes in such patients, the pulmonary lesion will often show better progress toward healing. Prior to the use of antituberculosis drugs such operations could not have been undertaken. However, now that pulmonary lesions can be kept under control by drug therapy, diseased nodes may be removed with safety.[22]

Those patients who show clinical evidence of extra-pulmonary tuberculosis or generalized tuberculosis must be treated with prolonged antituberculosis chemotherapy and bed rest; their cervical glandular tuberculosis is of secondary importance. Surgical procedures in the early stages are limited to those individuals in whom the nodes become fluctuant and are in danger of rupture; these should be aspirated or drained with subsequent use of streptokinase-streptodornase. After the extra-pulmonary lesions are well stabilized following at least 6 to 12 months of therapy, the cervical node tubercu-

losis may be evaluated. If a large mass of nodes remains, surgery may be considered at this time.

Removal of Tonsils and Adenoids

In those patients whose involved cervical node is one which may drain the tonsils or adenoids, special consideration should be given to tonsillectomy and adenoidectomy.[4, 7, 8] These patients include those in whom the involved node is in the jugular-mandibular group, the posterior triangle-lateral cervical group or the pre-auricular group. Removal of the tonsils and adenoids is recommended for two reasons: the tonsils and adenoids may be a focus for tuberculous infection; or they may also be a focus for a low grade bacterial or viral infection, and the node may tend to subside following their removal.

X-ray Therapy

The use of x-rays in the treatment of tuberculous cervical adenitis should be reserved as an adjunct to chemotherapy and surgery.[23] Roentgen ray therapy does not kill tubercle bacilli, and it does not cause the disappearance of either a cold abscess or caseation necrosis.[18] The chief value of x-ray therapy is the stimulation of fibrosis. Although a few radiologists have been enthusiastic about the results of x-ray therapy used as the principal therapy,[24] most workers in the field have reported an experience which was far from spectacular. X-ray therapy is primarily indicated in those cases where there is a persistent sinus tract. In most patients surgery should first be tried to remove such a sinus tract; however, if surgery has been ineffective or if the tuberculosis is so extensive that surgery is contraindicated, roentgen ray therapy should be used. X-ray therapy is also used in certain patients with multiple involved nodes. The most commonly recommended dosage is between 900 r and 1,000 r per field. One suggested plan of treatment is the use of 100 r every 10 days for 10 doses.[4]

Occasional complications have been reported after the use of x-ray therapy for tuberculous adenitis; these include: 1) breakdown of tuberculous nodes;[25] 2) extensive thickening and scarring of skin;[2] and 3) activation and spread of tuberculosis in other parts of the body.[18] It should be stated, however, that such complications are rare in properly treated patients. It is probable that in some instances the x-ray treatment which was given has been erroneously blamed for events which might have occurred even in the absence of such treatment.

THE PROBLEM OF EQUIVOCAL DIAGNOSIS

Those patients whose diagnostic studies are so equivocal that the presence of a tuberculous infection is uncertain require a different plan of ther-

apy. After the initial two to three week course of penicillin combined with a broad spectrum antibiotic, a decision should be made concerning the removal of the patient's tonsils and adenoids. This operation should be performed if the node affected is one which may drain these organs.[7] Following the removal of the tonsils and adenoids, the patient is observed for another three weeks to determine if there is any improvement in the affected node. If there is no significant improvement, the patient should have a biopsy performed, with removal of the entire node. Penicillin and a broad spectrum antibiotic, which have been previously initiated, should be continued into the postoperative period for several weeks to guard against secondary infection. Whenever there is a strong probability that the patient will prove to have tuberculous cervical adenitis, it is recommended that isoniazid be started about 5 days before the biopsy.[20] This drug should be continued until the results of pathological study of the biopsy specimen have been determined. If the diagnosis of tuberculous cervical adenitis is definitely eliminated, isoniazid should be discontinued. However, in those patients in whom a diagnosis of tuberculous cervical adenitis is established by pathological examination, para-aminosalicylic acid should be added. Therapy with both this drug and isoniazid should be maintained for a minimum of one year.

There is some possibility that the use of an antituberculous drug for several days prior to the biopsy may subsequently interfere with obtaining positive cultures of tubercle bacilli. However, the biopsy may be performed with a greater degree of safety for the patient if the antituberculosis drug is started before the surgical procedure is performed; the use of isoniazid will help prevent the possibility of any spread of tubercle bacilli during the operation. Furthermore in most cases there will be no interference in the obtaining of positive cultures despite the use of this drug.

SUMMARY

In summary, tuberculous cervical adenitis is the most common form of peripheral tuberculous adenitis. Nodes infected with tuberculosis pass through an initial exudative stage. They then undergo varying degrees of tubercle formation, caseation, fibrosis and calcification. Healing of such a node may occur, but in many cases viable organisms may remain for many years. Treatment of a tuberculous cervical node varies with whether the patient has active tuberculosis limited to the node or whether there is also active tuberculosis in the lungs or elsewhere in the body. Those patients who have only tuberculosis of the cervical nodes should be treated with antituberculosis chemotherapy for a minimum of one year. This should be combined with surgical excision if the affected node has not completely subsided after three to six months of treatment. Removal of the tonsils and

adenoids should be done if the node involved is one which drains these organs. Whenever biopsy is required a tetracycline drug and penicillin should be given to prevent secondary infection. If a tuberculous node becomes fluctuant, some form of drainage must be done promptly. Patients with an active pulmonary lesion must be treated with general care and antituberculosis chemotherapy until the pulmonary lesion is quiescent; removal of the tuberculous node may then be performed. Patients with extra-pulmonary or generalized tuberculosis require long periods of treatment with isoniazid and para-aminosalicylic acid together with bed rest. After the extra-pulmonary lesions are well stabilized, surgical excision of the node may be considered. Roentgen rays should be used as adjunctive therapy in those patients with persistent sinus tracts and in those patients in whom surgery is contraindicated.

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Aphasia and Deafness in Children—Some Aspects of Differential Diagnosis

EDNA K. MONSEES*

In the years that have followed World War II, there have been tremendous advances in research and invention that have shed new light on problems of hearing and deafness. As a result we have had to re-examine many of the old maxims about deaf children, and we are beginning to suspect that some of them may not be true.

One of the statements, for example, that has long been accepted as fact is that the ability to communicate orally cannot be achieved by all deaf children—not even by certain deaf children who have normal mentality and good emotional adjustment. That is, it has been stated that a percentage of otherwise normal deaf children cannot learn to read lips and to talk. Many reasons have been put forth for this inability, and the one most often heard is that lip reading is an art, and its achievement is therefore dependent on a certain amount of talent. Some deaf children seem to have a flair for lip reading, and others just cannot get the knack of it at all.

Recent developments, however, seem to be pointing to a need for reevaluation of this point of view. It may be that the children who cannot learn to lip-read and to talk may actually be suffering from a handicap that is not

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primarily deafness at all—or that they may be children handicapped with both hearing and language disabilities. They may be aphasic rather than deaf, or perhaps aphasic *and* deaf. Since aphasic children cannot learn to communicate by the teaching methods that succeed with deaf children, it is obviously important to be able to make accurate differential diagnosis between children who are handicapped primarily by a loss of auditory acuity and those who have the additional handicapping language disorder of aphasia.

The purpose of this paper is to discuss some of the problems of making a differential diagnosis between deafness and aphasia, and in formulating the educational program for children handicapped by these conditions.

Aphasia is the inability to use and/or understand spoken language, and is the result of damage or defect in the central nervous system. Aphasia may not be given as the diagnosis when the language deficit can be attributed to mental deficiency, emotional disorder, or deafness alone. In the actual clinical situation, however, it is often not an either-or matter; that is, it is not a question of differentiating between deafness and aphasia, for example, but rather the problem of determining whether the failure to develop speech is the result of deafness alone or of aphasia alone, or a combination of deafness and aphasia.

There are two major types of aphasia: receptive or sensory aphasia, and expressive or motor aphasia. In the latter, motor aphasia, the child understands the speech of others but lacks expressive speech. In motor aphasia deafness can usually be ruled out readily. In sensory aphasia the child lacks understanding of spoken language and for this reason is also unable to use speech for communication. It is in receptive or sensory aphasia that the question of hearing impairment is most likely to arise.

It is not always possible to obtain reliable hearing tests in children with receptive aphasia. Standard audiometric procedures require the subject to indicate by some agreed upon signal whether or not he hears the various test tones. Such procedures imply the ability to recognize and respond appropriately to auditory stimuli. Receptive aphasia by definition involves the inability to recognize and utilize auditory symbols in the form of words, and it would seem reasonable to assume that there might also be some disturbance in the ability to react appropriately to isolated pure tone stimuli as well. Our experience confirms this assumption—children with receptive aphasia are often unable to give consistent, reliable responses in standard pure tone audiometry.

Hearing tests using electrodermal responses to the usual pure tone audiometric stimuli have been developed in the past 10 years. In this test situation the subject is not required to give any overt voluntary responses to the test tones. This test measures the sensitivity of the organism to the test

tones, and does not measure the subject's ability to recognize or respond to sound, which is the basis of standard audiometry. The two testing procedures measure two quite different aspects of what is commonly known as "hearing." It is not surprising, therefore, to find that the thresholds shown for a given subject tested by standard audiometric techniques and by electrodermal response audiometry are often widely disparate.

In the diagnostic clinic we are finding many children who are brought in because of lack of speech and suspected hearing loss, and who show a severe loss of hearing when tested by standard audiometry and normal thresholds when tested by electrodermal response. We also see many children who are too young to co-operate in standard audiometry and who appear to be profoundly deaf in their responses to environmental sounds and in the informal screening tests, but who show normal thresholds in the electrodermal response test. These seeming discrepancies are not real discrepancies, but rather it can be said that the two types of tests provide us with separate sets of facts about the child's hearing. When the child shows abnormal responses to environmental sound and abnormal thresholds on standard audiometry along with normal thresholds in electrodermal audiometry, we conclude that the auditory disorder is in the central nervous system rather than in the end organ of hearing.

The terms "central auditory disorder" and "central language disorder, or aphasia" are not synonyms. In some cases of central auditory disorder there seems to be largely a loudness factor involved, so that when the child is provided with amplification through a hearing aid, and auditory and speech training, he is able to learn to lip-read and talk. The term "aphasia" implies an additional language disability other than the purely auditory.

Skilled observation of the child in the clinic added to the observations of the parents, and often a longer period of diagnostic teaching and evaluation are necessary in order to obtain a fair appraisal of a child's actual hearing. The child's reaction to amplified sound in the form of a hearing aid often is an additional index to his hearing problem. The experience of most clinical workers indicates that children with impaired hearing enjoy the experience of hearing through a hearing aid. Often with no period of "breaking in" or with a very short period, young children who need hearing aids will wear them without complaint, and, in fact, will accept them with obvious eagerness and delight. In our work at Children's Hospital, we usually start out with the assumption that the problem with a child who does not respond normally to environmental sounds and to the speech of others is simply one of impaired hearing. Where we are unable to obtain an audiogram by standard audiometry because the child is too young to co-operate, or when we obtain an audiogram which our observations lead us to believe is not reliable, we prescribe a hearing aid for the child on an experimental basis. We have

a number of used hearing aids in good working order which we lend to the family during this trial period. Where the child refuses to wear the aid after a reasonable length of time, we conclude that the aid cannot be doing much good, and we discontinue its use until after a longer period of training and observation. If there is any possibility that amplification of sound will help a child in his task of learning oral communication, we prescribe a hearing aid and try to get him to accept it. Parenthetically, where the thresholds obtained by electrodermal response audiometry are normal, we use mild or moderate gain hearing aids to prevent the possibility of cochlear damage resulting from intense and sustained noise.

Thus, when we have a child in the clinic who does not talk and does not understand the speech of others, we use all of the technical equipment and experience at our disposal in the attempt to determine the state of his hearing. Sometimes we are able to conclude that the child has normal auditory acuity. In such cases, where mental deficiency or emotional disorder are not severe enough to have caused the condition, and particularly where we have a history of disease or accident that might have caused damage in the central nervous system, we may venture at least a tentative diagnosis of sensory or receptive aphasia. Sometimes, however, we may conclude that the child has a true hearing loss or lack of auditory acuity—or, to use rather loose terms that mean different things to different people, we might conclude that the child is "deaf" or "hard of hearing." When we have reached this conclusion, we have unfortunately not finished the diagnostic job nor arrived at any conclusions about the type of educational program that should be provided for the child.

It is generally true that the hearing-handicapped children who have the most residual hearing and who are able to develop good auditory discrimination are the ones who will be able to achieve the greatest ease in oral communication. However, experience has shown that the degree of hearing loss is by no means the only important factor in predicting the deaf child's ability to learn to lip-read and to talk. Just as we know that there are children with normal hearing who cannot use oral communication because of aphasia or aphasoid language disability, we must assume that there may also be children with impaired hearing who also have aphasia. In the diagnostic process we must always be aware of the possibility of dual or multiple handicaps.

We believe that deaf children of average or even somewhat below average intelligence, even those with profound hearing loss, can achieve satisfactory oral communication ability when provided with a hearing aid, auditory training and the type of instruction conventionally used in teaching deaf children, providing they do not also have an aphasoid language disability. The corollary of this statement is that where an aphasoid language disabil-

ity exists, the deaf child will not learn to communicate by the methods used in teaching deaf children, no matter how slight or severe the hearing impairment.

The diagnostic problem accordingly becomes that of determining which of the children who have been diagnosed as deaf or hard of hearing have a coexisting aphasia. The problem then is that of identifying the aphasic condition. Some writers believe that the aphasic child has certain behavioral characteristics that distinguish him from the child who is only deaf. They say, for example, that the deaf child uses gestures and the aphasic child does not, or that the deaf child is consistent in his failure to respond to environmental sounds while the aphasic child is not. Others have said that the traits considered generally descriptive of brain-injured children are characteristic of all aphasic children. Poor motor co-ordination has also been said to be typical of aphasic children. Other writers—and our own experience leads us to agreement with this group—indicate that there are no such behavioral and motor characteristics that differentiate the purely deaf children from the deaf-aphasic child. Some aphasic children exhibit these characteristics and others do not.

While some research has been done and is in progress in the attempt to correlate certain psychometric findings with congenital aphasia, to date this has not been productive of any major differentiating sign posts. Neurological examination and electroencephalography have also proved to be nondifferentiating.

The speech and language examination itself in the young child before training is initiated is not diagnostic between deafness alone and deafness accompanied by aphasia. McGinnis, Kleffner, and Goldstein, in an article on "Teaching Aphasic Children" in the Volta Review, indicated that their experience has led them to classify the vocalizations of children with receptive aphasia into four categories, as follows: 1) There may be little or no vocalization; 2) There may be jabber, chatter, or jargon; 3) There may be echolalia; or 4) There may be appropriate use of a limited number of words or phrases; the child may or may not understand these words or phrases when spoken to him. Except for echolalia, which is not possible in the presence of deafness, the other categories could be applied with equal accuracy in referring to the vocalizations of either deaf or aphasic children.

In the last analysis, at this stage of our knowledge at least, the determination as to which children are purely deaf and which are both deaf and aphasic lies in the child's response to attempts to teach him to lip-read and talk. In the related area of reading disability, the child with complete or partial reading disability may be differentiated from the child with no reading disability by his response to reading instruction—the child with this disability will not learn by the teaching methods by which the majority of children

learn to read. In like manner the child with a disability in oral communication, the aphasic deaf child, will fail to learn by the teaching methods which are successful with the majority of deaf children.

An analysis of the methods of teaching oral communication to deaf children, on the one hand, and the methods used for teaching children with receptive aphasia, on the other, provide us with some insight into the nature of the aphasic condition itself.

The usual methods of teaching lip reading and speech to deaf children range from the "natural language" method described by Dr. Mildred Groht in her recent book, to the more structured systems such as those centered about the Fitzgerald Key. Different as these methods may seem to be at first glance, they have certain common characteristics. They involve talking to the child in the expectation that by frequent repetition he will learn to recognize some speech through lip reading and will subsequently be able to produce some speech that is intelligible although perhaps poorly articulated at first. These methods assume that the child will develop considerable understanding of speech (lip reading) before he will be able to produce speech, and of course this means understanding of the stuff that speech is made of—words, phrases, and sentences. Reading goes along with the development of lip reading after the child has reached the age of about 5 or 6. Writing is introduced much later in the process. Phonics may be used later, and then largely in terms of improving articulation and reading skills.

Since these methods succeed with many deaf children, the question arises as to why they are not successful with all deaf children—or what is there about the teaching method or the child that prevents successful learning. Orton, writing in 1937, characterized receptive aphasia as follows (he called the condition "auditory aphasia" or "word deafness," however):

> While these children show many errors of a wide variety of kinds it is clear that their difficulty is in the recalling of words previously heard, for the purpose of recognizing them when heard again or for use in speech, and that one of the outstanding obstacles to such recall is remembering all of the sounds in a word and these sounds in their proper order. . . . It is the recall of sounds in proper temporal sequence which seems to be at fault.

Hardy has expressed this as follows:

> What is most obvious is that they cannot naturally listen, understand, store, and recall symbolic structures involving a time order and stress pattern.

Aphasia seems to be a disorder involving the time factor primarily—in order to understand speech through hearing or lip reading, the child must recognize transient symbols as they pass by and he must remember the sequence of these sounds or lip movements in order to recognize a series of such sounds or lip movements as a word or phrase.

Working in a school for aphasic children which is part of the Central Institute of the Deaf in St. Louis, Miss Mildred McGinnis and her associates about 25 years ago developed a method for teaching aphasic children. We have adopted this method, with some modifications, in our Children's Hospital school for children with severe hearing and speech disorders. The method has produced remarkable results with children who failed to learn when taught as deaf children. The essential features of the method are as follows: We start with the sound elements instead of words and phrases. The child is taught to recognize the letter symbols and to read the sounds the letters stand for. Then two or three sounds are put together to form a word. The word is written in two colors by the teacher after a structured drill leading up to the word. The child reads the sounds in sequence and then says the sequence from memory. Then the word is associated with a picture of the object it represents. The child traces the word on the blackboard —or copies it if he is able. He then watches the teacher's lips as she says the word broken into sounds and then smoothed; he repeats the word in the same manner, finds the picture, matches it to the written word on the board, and reads it aloud. Then the child listens while the teacher says the word, broken up and then smoothed directly into his ear, or behind him if he is using a hearing aid; the child repeats the word, finds the picture, matches it to the written form and reads it aloud. Finally the child recalls the word from the picture alone and later writes the word from memory. All sensory channels are stressed for the purpose of achieving memory of the sounds that go into a word and these sounds in proper sequence. There is the visual reinforcement of the word written in colors with a change of color signalling a change of sound; visual in the form of emphasis on reading the lip movements; kinesthetic through the requirement of precise articulation of each sound and through the experience of tracing, copying, and writing the words; auditory through the presentation of the word through this channel in isolation. After a substantial number of nouns have been learned, the building of sentences and more complex language forms begins. When sufficient language has been mastered by the child, the lessons are based upon the regular academic school subjects to provide a well rounded educational program and to prepare for the child's transfer into a regular school.

To summarize: In the diagnostic clinic we see many children who do not talk and who cannot understand spoken language. Where this condition is not due to mental deficiency, emotional disturbance, or deafness, and particularly where the history shows some disease or accident that might have caused central nervous system damage, we may make a tentative diagnosis of receptive aphasia which will be confirmed or revised after a period of diagnostic teaching. We must always be aware of the possibility of multiple handicaps, and aphasia may exist in combination with other handicaps.

The problem often involves determining which deaf children have deafness as their only handicap and which children are both deaf and aphasic. Audiometric, neurological, psychometric and other examinations are often undifferentiating in individual cases. The final determination of which deaf children are also aphasic lies in the child's response to teaching. The deaf child will learn by the methods currently used in teaching oral communicaton to the deaf. The aphasic deaf child will fail to learn by these methods and requires an altogether different teaching procedure. The importance of early diagnosis is obvious: to prevent the waste of time involved in trying to teach an aphasic deaf childs by methods entirely unsuited to his needs, with all that this failure may mean to the child, his parents, and the teacher.

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The Serious Implications of Anaphylactoid Purpura

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Anaphylactoid purpura is generally recognized today as a diffuse allergic (or hypersensitive) vasculitis, the principal clinical manifestations of which include: 1) urticarial and subsequent purpuric skin lesions, usually involving the extremities and the face, but sparing the trunk; 2) rheumatoid-like joint symptoms (more commonly referred to as Schönlein's purpura); and 3) gastrointestinal symptoms of colicky pain, and occasionally marked rigidity of the abdominal wall (known as Henoch's purpura).

The etiology of this disease still remains a mystery, although most workers in the field are of the opinion that an allergic response to various allergens is the basis for the pathological vascular changes. An upper respiratory tract infection frequently precedes the onset of the disease. Some workers have made an attempt to classify this disease among the so-called "collagen diseases" because of the striking similarities that exist between anaphylactoid purpura and glomerulonephritis, serum sickness and rheumatoid arthritis.[1]

Basically, anaphylactoid purpura is a nonthrombocytopenic purpura, in which the defect is presumed to lie in the smaller blood vessels—the arterioles. The permeability of these vessels is increased, and there is either exudation of blood, producing purpura or visceral hemorrhages, or exudation of plasma, producing urticaria or edema.[1]

The occurrence of anaphylactoid purpura is uncommon in adults, and is most frequently seen in children, the highest incidence being in the age group between 4 and 7 years. Repeated attacks are common thereafter, but as the child becomes older, the disease becomes milder and usually disappears. Males are definitely more often affected than females; in some series the ratio has been noted to be as high as three to one.[2] Of interest is the recent study of this disease in Scotland by Lewis,[3] who found that the syndrome had a distinct spring and autumn peak, at which times it was also noted that the incidence of respiratory infections was at its greatest. In addition, it has also been reported that there was a significant number of cases of anaphylactoid purpura preceded by a respiratory infection in which the beta hemolytic streptococcus was present on throat culture. Gairdner[4] called attention to the close relationship of anaphylactoid purpura to acute nephritis and to rheumatic fever, both of which are now accepted as being associated with a preceding group A beta hemolytic streptococcal infection. However, Bywaters, Isdale, and Kempton,[5] in their rather complete review of 64 cases of anaphylactoid purpura, arrived at the conclusion that although there are

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close clinical and pathological similarities between anaphylactoid purpura and nephritis and rheumatic fever there is no clear evidence of infection with group A beta hemolytic streptococci as an *invariable* causative factor.

Anaphylactoid purpura is generally considered to have a good prognosis unless complications such as nephritis, intussusception, intestinal perforation or cerebral hemorrhage occur. This paper is an attempt to focus attention on the complications of anaphylactoid purpura, and to emphasize the serious implications of these complications.

Renal Complications

The most serious of all the complications is the occurrence of glomerulonephritis. This may appear at any time in the course of the disease. According to Derham and Rogerson,[6] Henoch, as early as 1899, made the statement that nephritis was a frequent complication of anaphylactoid purpura, and might be so severe as to cause anasarca and death. Since that time, many series of cases have been reported in which the incidence of nephritis was indeed high; these reports also emphasize that death from renal failure is entirely possible.

Typical urine abnormalities which occur are microscopic hematuria and occasional proteinuria. At times, however, marked disturbance of renal function has occurred. Uremia and hypertension with ensuing death may rarely occur.[7] Signs of the nephrotic syndrome may occur in the course of anaphylactoid purpura.[8]

Gairdner[4], in his classic review of this syndrome in 1947, showed that this type of nephritis, just as acute nephritis of any other etiology, may go on either to recovery, to latent nephritis, to chronic nephritis, or to rapidly developing renal failure and death. In his series of cases he found that when macroscopic hematuria was present it often persisted throughout all stages of the nephritis, so that a patient in the subacute or chronic stage of nephritis might still have obvious hematuria. Apart from this feature, he concluded, the renal disease seemed to differ in no way from the usual forms of glomerulonephritis.

Pathologic changes in the kidneys of those patients who die and on whom necropsy studies are done are interesting. The intertubular tissues contain numerous foci of small hemorrhages and collections of polymorphonuclear leukocytes, eosinophils, lymphocytes and plasma cells. These particular foci are quite similar to the inflammatory foci seen in the cutaneous lesions.

The kidney may be involved at any time during the evolution of anaphylactoid purpura; the resultant acute nephritis is usually followed by apparent complete recovery. However, repeated urine examinations over a period of time may reveal exacerbations of the renal disease at varying intervals, suggesting a continuing or intermittent pathologic process in the kidney.

The usual urinary findings in these particular cases are gross or microscopic hematuria, casts and proteinuria.[9]

Another interesting finding was demonstrated in a study of 26 patients with anaphylactoid purpura who were followed for periods of from 2 to 15 years. Ten of these patients had abnormal urinary findings, but only 6 of the 10 had proteinuria or hematuria during the acute stage of the disease.[6] It is seen, therefore, that nephritis may occur as a late sequel in some cases, even when urine examinations show no abnormality during the acute stage. It is for this reason that some authors have postulated that anaphylactoid purpura might be considered as a possible latent cause of nephritis in older children and adults. Indeed, one wonders whether certain cases of so-called "nephritis of unknown etiology" are not merely cases of latent nephritis caused by anaphylactoid purpura.

Derham and Rogerson[6] have attempted to differentiate the nephritis that occurs with anaphylactoid purpura from true nephritis. It is definitely known that renal damage, as shown by hematuria, proteinuria or both, does occur, but it has not been established that these findings constitute acute glomerulonephritis. The authors point out that in anaphylactoid purpura facial edema and hypertension are not commonly found, that nitrogen retention is rare, and that the erythrocyte sedimentation rate tends to return to normal within a short time despite continued hematuria. In addition, they contend, the serum complement is normal in anaphylactoid purpura, and it is markedly reduced in true nephritis.

In a recently published paper, Vernier et al.[10] report on eight children with anaphylactoid purpura during the acute and convalescing stages (in one case, five years later) in whom renal biopsies were performed. It was found that the basic morphologic abnormality appeared similar in all 8 cases, and consisted of segmental glomerular proliferation and occlusion of capillaries by Schiff-positive fibrinoid material in the acute stage. Chronologically older lesions appeared as segmental areas of organized hyaline material or as frank segmental scars of fibrous tissue. These authors contend that most present-day morphologic studies indicate that the renal lesion of anaphylactoid purpura is *not* related closely to the renal lesion of acute glomerulonephritis. In fact, immunologic studies support the theory of different etiology: High levels of anti-streptococcal antibodies are found in patients with recent acute glomerulonephritis, but such antibody titers in patients with anaphylactoid purpura are usually normal. In conclusion, these authors suggest that the nephritis of anaphylactoid purpura is a diffuse vascular disease, resulting in pathologic renal lesions which resemble the renal lesion of disseminated lupus erythematosus.

In any event, it becomes obvious that in patients who have several recurrences of anaphylactoid purpura accompanied by evidence of nephritis the

ultimate prognosis is usually less favorable than in patients with uncomplicated recurrent anaphylactoid purpura.

Gastrointestinal Complications

Gastrointestinal symptoms in anaphylactoid purpura are variable and frequently precede the skin eruption. There is usually colicky abdominal pain, at times quite severe, but not definitely localized. There may be vomiting of varying degrees of severity, and at times blood may be found in the stools. Thus it is apparent that many cases of anaphylactoid purpura may be confused with other intra-abdominal conditions such as appendicitis, regional ileitis and intussusception.

Esposito[11] confirmed the previously reported finding that one can demonstrate by means of a barium meal pathologic small intestinal changes before the onset of the skin rash. In addition, he found that the small intestine reverted to normal after the abdominal symptoms had disappeared. He reported the following radiologic changes occurring in the small intestine of patients with abdominal symptoms of anaphylactoid purpura: 1) The normal mucosal patterns in the duodenum and jejunum are altered; there is obliteration of the mucosal folds, and dilatation and segmentation of the ileum. 2) There is pooling of the barium in the stomach. 3) There are small mucosal ulcerations, as shown by the presence of small detached barium particles. 4) There is marked local hypomotility.

Brust[12] states that in uncomplicated anaphylactoid purpura with abdominal symptoms there is an urticarial, serosanguinous effusion into the intestinal wall which produces the colicky pain. (Some authors[13] have referred to this particular phase of abdominal purpura as "purpura abdominalis" because of the similarity of the intestinal mucosal changes to the typical skin lesions.) The affected segment of the small intestine appears red, edematous, firm and indurated. Two major complications may then ensue, both of which are rare: intussusception and perforation.

Many who are unfamiliar with the peculiarities of anaphylactoid purpura might interpret the abdominal symptoms as warranting an exploratory laparotomy. Muldoon et al.[14] list certain considerations which put the surgeon on guard in an attempt to aid him in interpreting the abdominal symptoms correctly: 1) absence of an evident cause for the abdominal symptoms; 2) associated skin and joint involvement; 3) shifting of the pain from one part of the abdomen to another; 4) change in the character of the pain during different attacks; and 5) lack of abdominal rigidity and muscle spasm in keeping with the severity of the pain or the degree of tenderness present.

However, these cases may at times be complicated by intussusception, and one should definitely not hesitate to operate in the presence of a palpable abdominal mass. Steinhardt and Jonas[15] feel strongly that, even in the *absence* of a palpable mass, should the melena and abdominal pain be severe,

operation is urgently indicated, since previous work has shown that a gangrenous intussusception can be present without a palpable mass. The high percentage of resections of the diseased intestine in the past, they believe, results from much delay in the decision to operate. These authors feel that the danger of surgery in the presence of purpura is slight compared to the danger implicit in delay when intussusception is present.

In the chronic form of anaphylactoid purpura, the gastrointestinal picture is one of recurrent acute episodes often occurring concomitantly with joint and/or skin manifestations. These attacks are usually brief and self-limited, but may also occur in rapid succession and be of sufficient duration to cause total incapacity. Often these patients become distraught over the repeated unexplained abdominal crises, and more often than not they are subjected to unnecessary exploratory surgery. These shortcomings can be minimized only by developing an awareness of this disease in both its acute and chronic aspects.[8]

Cardiac Complications

MacGregor and Vallance-Owen[16] have reported 3 cases in which there was clinical and electrocardiographic evidence of myocardial damage during attacks of anaphylactoid purpura. All 3 patients were elderly, but it would not be difficult to conceive of the occurrence of cardiac damage in children. Clinically, the manifestations were remarkably alike in all these cases. In two of the patients sudden symptoms and signs of cardiac failure coincided with the onset of purpura. These patients had temporary atrial fibrillation at first; one of them also developed nodal rhythm with paroxysmal heart block. The third patient developed a wandering pacemaker during a recurrence of purpura. Electrocardiographically, there were sequential changes in the ST segments which returned to normal within from a few days to a few weeks. Serial electrocardiograms, the authors contend, did not support the suggestion that the patient's illness had simply provoked an attack of coronary thrombosis. The electrocardiographic changes also resembled those which are sometimes found in acute nephritis; in one of these cases, however, there was no evidence of renal involvement.

These authors feel that the heart may be affected as often as the kidneys during anaphylactoid purpura since the vascular lesion is widespread and the affected vessels are susceptible to mechanical trauma. The only clinical manifestation which might be expected in the average case is a temporary alteration in the electrocardiogram. Inasmuch as electrocardiography is not done routinely in children, these same authors emphasize that cardiac involvement may be easily overlooked because it is evidently rarely serious. However, it becomes important when the disease is unusually severe or prolonged, or when the heart is already affected by degenerative disease. It is for this reason that they recommend doing an electrocardiogram at an

early stage in every severe case. Should a cardiac lesion then be found, steroid therapy is indicated, since this can perhaps prevent the development of more serious changes.

Neurologic Complications

Not much has appeared in the literature regarding the neurologic complications of anaphylactoid purpura. However, Lewis and Philpott[17] reported 3 cases of children who during the course of an attack of anaphylactoid purpura developed the following symptoms: 1) convulsions, presumably hypertensive, in the absence of renal involvement; 2) subarachnoid hemorrhage; and 3) intracerebral hemorrhage due to arteriolitis. The occurrence of widespread arteriolitis has been noted in various necropsy reports, and it is not inconceivable that the same pathologic process is responsible for the cerebral symptoms.[4]

Death will occur should the cerebral damage be great enough, but in several cases remission of the neurologic changes has been known to occur. Thus, transient hemiplegia, transient paresis and chorea have all been reported.[15, 17]

Anaphylactoid Purpura and Tuberculosis

For some time anaphylactoid purpura has been known to sometimes accompany tuberculosis. It had originally been noted to occur with significant frequency in patients with terminal tuberculosis, and for this reason the onset of purpura in patients with tuberculosis was considered an ominous sign.[18] However, many subsequent studies have not supported this contention. Rather, a very high proportion of cases was found in disseminated tuberculosis, and a few cases were found following splenic tuberculosis.[18] Pathologically, capillary damage has been implicated, but it is also thought that bone marrow involvement is the factor responsible for the purpura.

Because of this peculiar association of purpura and tuberculosis, Dalgleish and Ansell[19] have stressed the importance of searching for a tuberculosis focus in the presence of an unexplained purpura.

Other Complications

Other complications that may arise from anaphylactoid purpura are: 1) ocular hemorrhages, sometimes so severe as to necessitate enucleation, and 2) purpura gangrenosa, in which the skin lesions become necrotic and at times require excision and plastic repair.[20, 21]

SUMMARY

The various complications of anaphylactoid purpura are discussed. Renal complications may not be present during the acute stage of the disease, so that a thorough search must be made in so-called "nephritis of unknown etiology." Anaphylactoid purpura may also cause chronic nephritis and, rarely, renal failure and death.

Gastrointestinal complications of importance are intussusception and perforation. The importance of familiarity with all the peculiarities of this disease is stressed in an attempt to avoid unnecessary exploratory abdominal surgery.

Cardiac complications include transitory clinical and electrocardiographic changes. It is suggested that an electrocardiogram be done in all severe or unusually prolonged attacks of anaphylactoid purpura so that proper therapy may be instituted promptly.

Neurologic complications may be only transitory in nature or may be permanent and result in death.

The importance of searching for a tuberculous focus in all cases of unexplained purpura is mentioned.

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The Editor's Column

Shotgun Orders

We are all familiar with the term "shotgun prescription," a prescription composed of many therapeutic agents which may be effective in a wide range of disorders. A doctor writing such a prescription probably admits to himself he does not know what is wrong with his patient, but hopes that one of the ingredients might be a specific for the patient's ailment.

Not infrequently, when one examines hospital chart orders he sees a long list of "shotgun" orders—orders covering a wide variety of laboratory studies—some of which are entirely unrelated to the patient's condition. To me this indicates one of two things: Either the orders were written before a careful evaluation of the patient was made; or, after a careful evaluation of the patient, the doctor has not the vaguest idea of what the trouble may be, and so begins to "shoot wildly" with tests and examinations, hoping one might "hit the mark."

I am reminded of the saying of one of my professors in medical school: "It is a sorry doctor who has to sit down and play solitaire with a bunch of laboratory and special examination reports to arrive at the correct diagnosis." Instead, it is far better to order a few well chosen procedures which, after careful consideration of the patient, would seem most likely to aid in substantiating a diagnosis. Such laboratory tests or examinations should be performed only to confirm a well founded suspicion of a disorder arrived at by the physician after a very careful history has been taken and a careful physical examination made.

Every physician is probably guilty of the tendency to write "shotgun orders" at one time or another; since new laboratory procedures are constantly being discovered, it is a great temptation to "throw the laboratory at the patient." This not only tends to dull our own diagnostic ability, but adds to the cost of hospitalization. Someone has to pay, even in the case of the staff patient. It is also well to remember that "wild shooting" with "shot gun orders" can cost the patient his life. Not all diagnostic procedures are without danger.

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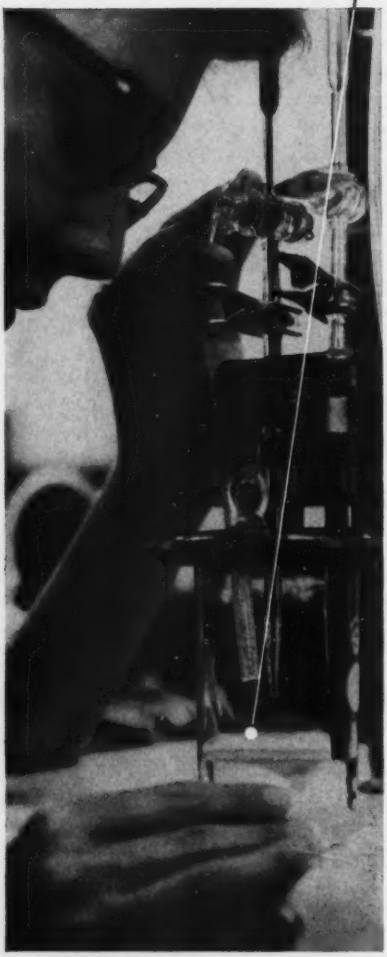

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